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MAOA, Early Experiences of Harsh Parenting, Irritable Opposition, and Bullying–Victimization: A Moderated Indirect-Effects Analysis

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Harsh parenting and child characteristics such as opposition and aggression have been found to relate to bullying, victimization, and bullying–victimization, yet not all children display equal vulnerability to harsh parenting. The monoamine oxidase A gene (*MAOA; low-activity* variant) may be a key vulnerability allele as it relates to aggression on experience of harsh parenting, and opposition in children, and may therefore be associated with children who become bullies and victims. Using multiple-informant data from 4,893 mother–child pairs participating in the Avon

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Longitudinal Study of Parents and Children (ALSPAC), we found that (a) harsh parenting increased subsequent victimization in boys, and the risk was higher for those with the low-activity allele; that (b) harsh parenting (mother reported) increased bullying, victimization, bullying–victimization (child self-reported) for boys but not for girls, via irritable opposition (mother and teacher reported); but that (c) this indirect effect was not moderated by *MAOA*. The results suggest that vulnerable boys who are treated harshly by their parents have increased victimization experiences, whereas irritable opposition appears related to bullying with and without victimization and related to victimization alone.

Parental maltreatment is associated with a wide spectrum of cognitive, emotional and behavioral problems in children (Ford et al., 2000). For example, children who have been maltreated or subjected to harsh parenting (i.e., frequently being shouted at, smacked, and told off) have been shown to behave more aggressively toward their peers (Dodge, Greenberg, & Malone, 2008). This aggression toward peers, sometimes also referred to as *bullying,* is thought due, in part, to poor socialization in homes where parents behave aggressively toward their children (Dodge & Pettit, 2003). Moreover, both child aggression (toward peers) and harsh parenting predict increased victimization by peers (Barker, Arseneault, Brendgen, Fontaine, & Maughan, 2008; Barker, Boivin, et al., 2008; Hosser, Raddatz, & Windzio, 2007). Hence, harsh parenting can influence the degree to which children bully their peers and are victimized by their peers.

As stated, one potential reason for the increased risk of becoming a bully or a victim after experiencing harsh parenting is that such parental behavior creates an interpersonal template for relating with peers that is not conducive to the development of normative and harmonious peer relationships (Dodge et al., 2003). This may foster the development of individual characteristics that predispose children to behave with reactive aggression toward peers as a result of interpersonal conflict (Ford et al., 2000). Reactive aggression can be defined as poorly modulated anger and irritability (e.g., Toblin, Schwartz, Hopmeyer Gorman, & Abou-ezzeddine, 2005), and high levels of reactive aggression has been found to exist in bullies, victims, and bully–victims (e.g., Camodeca, Goossens, Frits, Meerum Terwogt, & Schuengel, 2002; Salmivalli & Nieminen, 2002). Reactive aggression is akin to the *irritable opposition* dimension (e.g., being easily annoyed, engaging in temper tantrums, expressing anger) as each relates to increased aggression, being victimized, and harsh parenting (Barker & Salekin, 2012; Dodge, 2006; Ford et al., 2000; Pellegrini, Bartini, & Brooks, 1999; Schwartz, McFadyen-Ketchum, Dodge, Pettit, & Bates, 1998).

That said, not all children who experience harsh parenting are equally affected by this potential stressor—that is, some do not appear at risk for

increased aggression or irritable opposition following exposure to harsh parenting. The results of gene–environment studies indicate that some children are more sensitive to a toxic social environment than are others. More specifically, research has shown that the gene encoding the enzyme monoamine oxidase A (*MAOA; low-activity* allele) underlies biological stress-response systems and confers a vulnerability to harsh parental treatment—it moderates the relationship between parental maltreatment and later incidences of aggressive behaviors, including bullying and violence (Caspi et al., 2002; Kim-Cohen et al., 2006). The MAOA enzyme metabolizes a number of neurotransmitters—norepinephrine, epinephrine, serotonin, and dopamine (Caspi et al., 2002)—that are associated with multiple brain functions linked with stress regulation. The low-activity allele is linked with lower amounts of MAOA enzyme and thus higher amounts of circulating neurotransmitters—which in turn are associated with a hyperresponsive amygdala and hippocampus in reaction to emotional stress, alongside impaired performance of the regulatory prefrontal regions of the brain (Meyer-Lindenberg et al., 2006).

Of interest, individuals who carry the low-activity allele are also at risk for heightened impulsivity and reactive opposition (i.e., aggression linked to retaliatory irritability and emotional reactivity) on having experienced childhood physical abuse (Huang et al., 2004; Jaffe et al., 2005). Indeed, because the *MAOA* low-activity allele is also related to reactive aggression, low frustration tolerance, and irritability in children (Manuck, Flory, Ferrell, Mann, & Muldoon, 2000; Meyer-Lindenberg et al., 2006), this behavior may be appropriately labeled as an endophenotype of *MAOA*. More specifically, an endophenotype is a measure of an intermediate phenotype (i.e., irritable opposition) that is closer to the gene functioning compared to a more distal outcome (e.g., bullying). Hence, irritable opposition might explain an identified relationship between *MAOA* and bullying.

In addition, as stated earlier, harsh parenting predicts both bullying and victimization. Indeed, there is group of children who bully their peers while being victimized themselves, and they are sometimes referred to as aggressive victims (Pellegrini, Bartini, & Brooks, 1999), or bully–victims (e.g., Boulton & Smith, 1994). Moreover, higher levels of oppositional temperament are associated with greater risk to become bully–victims and also increase the likelihood to (solely) bully and be victimized (Barker & Salekin, 2012; Olweus, 1978). We posit that, when children have experienced harsh parenting, *MAOA* low-activity allele carriers will be at risk for bullying and bullying–victimization, via an increase in their irritable opposition, but no explicit hypothesis for victimization can be formulated based on the existing literature. More specifically, for the outcome

of victimization, our analyses are more exploratory—no risk allele is identified and no specific hypothesis is posited.

The current study, using the Avon Longitudinal Study of Parents and Children—a prospective epidemiological cohort sought to test for (a) the presence of a gene–environment interaction (G×E) for the *MAOA* low-activity allele and harsh parenting on bullying, victimization, and bullying–victimization; (b) an indirect-effects model, where harsh parenting increases bullying, victimization, and bullying–victimization via increased irritable oppositional behaviors; and (c) the degree to which the indirect effects (e.g., harsh parenting compared to bullying or bullying–victimization via irritable opposition) are stronger for *MAOA* risk allele *low-activity* carriers compared to carriers of the *high-activity* variant.

Method

Sample

The Avon Longitudinal Study of Children and Parents (ALSPAC) was established to understand how genetic and environmental characteristics influence health and development in parents and children. All pregnant women resident in a defined area in the South West of England, with an expected date of delivery between April 1, 1991, and December 31, 1992, were eligible and 13,761 women (contributing 13,867 pregnancies) were recruited. These women's cases have been followed over the last 19–22 years (Fraser et al., 2012). When compared with 1991 National Census Data, the ALSPAC sample was found to be similar to the UK population as a whole (Boyd et al., 2012). Ethical approval for the study was obtained from the ALSPAC Law and Ethics Committee and the local research ethics committees. More detailed information on ALSPAC is available at www.bris.ac.uk/alspac/.

Measures

Bullying and peer victimization measures, respectively (child reports), at age 8 were collected at the ALSPAC Child in Focus Clinics (see Schreier et al., 2009). The children responded how often (1 = *never* to 4 = *often*) they had experienced/engaged in the following: (a) had hit others/had been hit, (b) had stolen the belongings of others/had belongings stolen, (c) had called others names/had been called names, or (d) had threatened others/had been threatened. First, a measure of bullying was created by confirming the factor structure by using the items and saving the aforementioned factor scores ($\alpha = .53$). Second, in the same manner, a measure of peer victimization was also created ($\alpha = .61$). Third, a measure of bullying–victimization was

created by running a confirmatory factor analysis using the saved bullying and victimization factor scores as factor items ($\alpha = .61$). Because of the low alphas, we assessed internal reliability also through the goodness of fit in confirmatory factor analysis and found adequate fit for bullying ($\chi^2[2] = 0.057$, $p = 0.971$; comparative fit index [CFI] = .1.00, Tucker–Lewis index [TLI] = 1.00, root mean square error of approximation [RMSEA] = .00, 90% CI [.00–.00]), victimization ($\chi^2[2] = 0.379$, $p = 0.828$; CFI = .1.00, TLI = 1.00, RMSEA = .00, 90% CI [.00–.02]), and bullying–victimization ($\chi^2[2] = 0.032$, $p = 0.984$; CFI = .1.00, TLI = 1.00, RMSEA = .00, 90% CI [.00–.00]).

Harsh parenting between ages 2 and 4 was derived by using confirmatory factor analysis and saving factor scores. It was assessed by the mothers answering, "When you are at home with your child, how often do you do the following?" (a) shout at him/her and (b) slap him/her (for ages 2 and 4) and (c) tell him/her off (at age 4) ($\alpha = .76$). Response scale (reverse coded) was from 1 = *every day* to 5 = *rarely/never.*

Irritable opposition at age 8 was derived from the Development and Well-being Assessment (DAWBA), a well-validated measure, developed for the British Child Mental Health surveys (Meltzer, Gatward, Goodman, & Ford, 2000). The DAWBA assesses nine separate symptoms of oppositional defiant disorder (ODD). Each question was introduced to mothers and teachers with this question: "Over the last six months, and as compared with other children the same age, has s/he often . . . ?" followed by the specific clause. Following the lead of Stringaris and Goodman (2009), *irritable opposition* was defined by the following three symptoms: (a) has temper outbursts, (b) has been touchy or easily annoyed, and (c) has been angry or resentful. Items were coded on a 3-point scale where 1 = *no more than others,* 2 = *a little more than others,* and 3 = *a lot more than others*. Children were assigned a diagnosis only if their symptoms were causing significant distress or social impairment ($\alpha = .73$).

In addition to generating binary (yes/no) diagnostic indicators, DAWBA algorithms have been developed to generate six-level ordered–categorical measures of the probability of disorder for each of the individual items underlying the diagnoses, ranging from $<0.1\%$ to $>70\%$ (Goodman, Heiervang, Collishaw, & Goodman, 2011). The DAWBA has been evaluated in two large-scale national samples and has functioned well as ordered–categorical measures, showed dose–response associations with mental health service contacts, and showed very similar associations with potential risk factors as clinician-rated diagnoses (Goodman et al., 2011). Confirmatory factor analysis was employed to generate a factor score based on the three irritable opposition items.

MAOA genotypes—for this selected sample—were available for 2,506 boys and 2,387 girls. As the *MAOA* gene is located on the X chromosome, only females can carry two of the same alleles (*homozygotes*) and two different alleles (*heterozygotes*); males (*hemizygotes*) carry one allele only. *MAOA* 3.5- and 4-repeat alleles were coded *high activity* because enzyme expression has been found to be 2–10 times higher compared to 3-repeat alleles; the 2- and 5-repeat alleles were excluded from the analysis because their activity levels have not yet been established (Sabol, Hu, & Hamer, 1998). Non-White participants were excluded from the analysis, and males and females with at least one low-activity *MAOA* allele were coded as 1 and those without the allele were coded 0. The genotype frequencies for boys were L, 34.0%, and H, 66.0%; and for girls were LL, 12.4%, LH, 45.2%, and HH, 42.4% (where L stands for low-activity allele and H for high-activity allele). All genotypes conformed to Hardy–Weinberg equilibrium as reported by Enoch, Steer, Newman, Gibson, and Goldman (2010; see also Barnett, Xu, Heron, Goldman, & Jones, 2011).

Analysis

Analyses proceeded in four steps, each one completed by using separate models for boys and girls, respectively. In Step 1, the presence of a G×E for the *MAOA* low-activity allele and harsh parenting on bullying–victimization was tested by using linear regression.

In Step 2, an indirect-effects model was tested in which harsh parenting increases the risk for bullying, victimization, and bullying–victimization via increased irritable oppositional behaviors. Here, we tested for the presence of indirect effects rather than mediation. Indirect effects use the same calculations as mediation analyses, whereby the effect tested is the product term of the pathways that move from the initial predictor to the outcome via intermediary predictor variables. However, unlike mediation (Baron & Kenny, 1986), indirect effects do not require the presence of a direct effect between two variables in order to explore whether this association may be indirectly explained by a third intermediary variable (i.e., Collins, Graham, & Flaherty 1998; MacKinnon, 2000; Preacher & Hayes, 2008). This is particularly relevant for developmental research where predictive processes are likely to be distal (Shrout & Bolger, 2002).

In Step 3, we tested the degree to which this indirect effect (i.e., harsh parenting on bullying via irritable opposition) may be moderated by *MAOA*. In other words, we examined whether the strength of the indirect effect varied as a function of genotype. Figure 1 displays the model tested (using models for boys and girls, respectively)—that is, the presence of

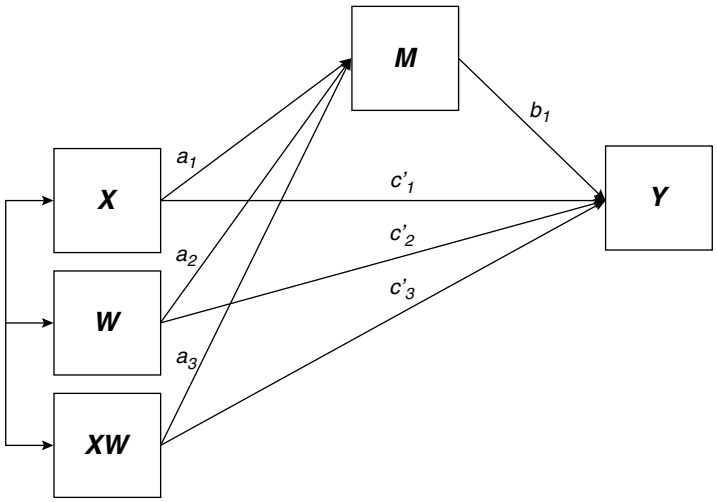

Figure 1. Moderated indirect-effects model (adapted from Model 2 in Preacher, Rucker, & Hayes, 2007). In analytic Step 3, *X* = harsh parenting, *M* = irritable opposition, *Y* = bullying–victimization, and the moderator (i.e., *W*) = monoamine oxidase A (MAOA). Here, paths a_1, a_2, and a_3 indicate indirect pathways linking main effects *X* and *W* and the interaction term *XW,* respectively, with *M;* in turn, the b_1 pathway links *M* with the outcome *Y*. Paths c'_1, c'_2, and c'_3 link *X, W,* and *XW,* respectively, with the outcome *Y*.

an indirect effect of *X* on *Y* through *M* (i.e., an indirect effect—refers to path a_3), for different conditional values of a moderator, *W* (see Preacher, Rucker, & Hayes, 2007), where *X* = harsh parenting, *M* = irritable opposition, *Y* = bullying, victimization, or bullying–victimization, and the moderator (i.e., *W*) = *MAOA*. In Figure 1, the paths a_1, a_2, and a_3 indicate indirect pathways linking main effects *X, W,* and the interaction term *XW,* respectively, with *M;* in turn, the path b_1 links *M* with the outcome *Y*. The paths c'_1, c'_2, and c'_3 link *X, W,* and *XW,* respectively, with the outcome *Y*. As standard errors underlying indirect effects (i.e., unstandardized coefficient product terms) are known to be skewed, we bootstrapped all indirect effects 10,000 times with bias-corrected 95% CIs.

All analyses were conducted in M*plus* version 6.21 (Muthén & Muthén, 1998–2010). To provide robust estimates and to account for missing values, maximum likelihood estimation with robust standard errors (MLR) was used. Individual model fit was determined through the comparative fit index and Tucker–Lewis index (CFI and TLI; acceptable fit $\geq$ 0.90) (Bentler & Bonett, 1980) and RMSEA (acceptable fit $\leq$ 0.08) (Browne & Cudeck,

1993). Model comparisons were conducted by using the Satorra–Bentler procedure for MLR.

Results

Descriptive Statistics

Descriptive statistics—means, standard deviations, and minimum and maximum factor score values of all study variables—are displayed in Table 1. As seen in Table 2, study variables were significantly positively correlated for both boys and girls: High levels of harsh parenting (age 2–4 years) were associated with high levels of bullying, victimization, and bullying–victimization (8 years) and high levels of irritable opposition (8 years), whereas high levels of irritable opposition (8 years) were associated with high levels of bullying, victimization, and bullying–victimization (8 years). We examined whether genotype was associated with harsh parenting, which would be indicative of a gene–environment correlation and needs to be ruled out prior to conducting G×E analyses. As depicted in Table 2, *MAOA* was not significantly correlated with any of the other study variables. The absence of an association between *MAOA* and harsh parenting was confirmed through mean comparisons of harsh parenting by

Table 1. Descriptive statistics for study variables by sex of the child

	Sex	
	Boys	Girls
MAOA (low-activity dominant) *N*	2,506	2,387
Harsh parenting (2–4 years) *M* (*SD*)	.07 (.80)	−.08 (.81)
Min. to max.	−2.45 to 1.97	−2.45 to 1.97
Bullying (8 years) *M* (*SD*)	.14 (.95)	−.14 (.53)
Min. to max.	−.31 to 7.11	−.31 to 7.07
Victimization (8 years) *M* (*SD*)	.09 (.85)	−.09 (.74)
Min. to max.	−.71 to 3.43	−.71 to 3.43
Bullying–victimization (8 years) *M* (*SD*)	.13 (.89)	−.14 (.61)
Min. to max.	−.58 to 5.87	−.58 to 5.85
Irritable opposition (8 years) *M* (*SD*)	.15 (2.23)	−.17 (1.76)
Min. to max.	−.99 to 10.66	−1.01 to 10.66

Note. All statistics are based on factor scores. MAOA = monoamine oxidase A; *M* = mean; *SD* = standard deviation; min. = minimum factor score value; max. = maximum factor score value.

Table 2. Bivariate correlations of the study variables by sex of the child

	MAOA (low-activity dominant)	Harsh (2–4 years)	Bul (8 years)	Vic (8 years)	Bul-vic (8 years)	Irrit (8 years)
MAOA (low-activity dominant)	—	−.03	−.02	.00	−.01	.00
Harsh (2–4 years)	.01	—	.04*	.08*	.08*	.16*
Bul (8 years)	.00	.10*	—	.37*	.77*	.07*
Vic (8 years)	.00	.11*	.47*	—	.88*	.08*
Bul-vic (8 years)	.00	.12*	.88*	.83*	—	.09*
Irrit (8 years)	.01	.14*	.14*	.14*	.16*	—

Note. Factor scores for each variable have been used. Boys' scores are below the diagonal; girls' scores above. *$p < 0.05$. MAOA = monoamine oxidase A; harsh = harsh parenting; irrit = irritable opposition; bul = bullying; vic = victimization; bul-vic = bullying–victimization.

genotype separately for boys and girls, which yielded no significant difference (not listed in the tables).

Step 1. Moderation of the Association Between Harsh Parenting and Bullying, Victimization, and Bullying–Victimization

The objective of Step 1 was to test for the presence of a G×E for the *MAOA* low-activity allele and harsh parenting on bullying, victimization, and bullying–victimization.

The results for boys showed that there was no significant G×E between harsh parenting and *MAOA* genotype for bullying, victimization, or bullying–victimization, although G×E approached statistical significance when victimization was the outcome measure ($\beta = -.08$, $p = .06$). Simple slope analyses showed that the association between harsh parenting and later victimization was stronger for low-activity allele carriers ($\beta = .14$, $p < .001$) and, while in the same direction, considerably smaller when the low-activity allele of *MAOA* was absent ($\beta = .06$, $p = .07$). These slopes are depicted in Figure 2. For both variants, the association between harsh parenting and victimization was positive.

For girls, there was no significant G×E between the *MAOA* variant and harsh parenting in relation to bullying, victimization, or bullying–victimization (see Table 3).

Step 2. Indirect-Effects Model

We next examined an indirect-effects model to test whether harsh parenting increases bullying, victimization, or bullying–victimization via increased

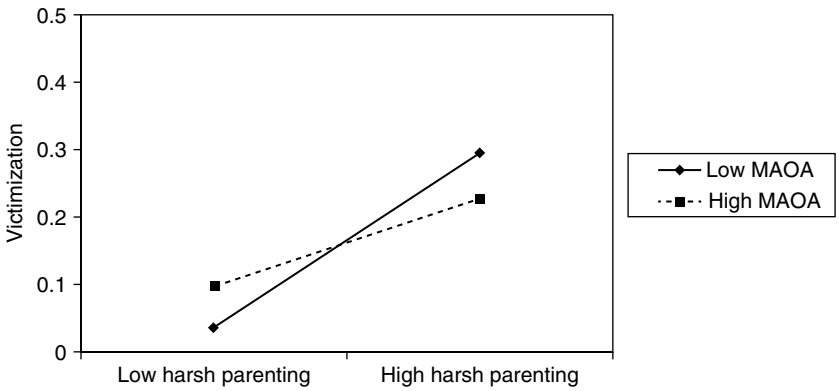

Figure 2. In boys, the association between harsh parenting and later victimization was significant for *low-activity* monoamine oxidase A (*MAOA*) allele carriers ($\beta = .14$, $p < .00$), whereas, for *high-activity-only* allele carriers, this interaction was not significant ($\beta = .06$, $p = .07$).

irritable oppositional behaviors. For boys, a significant indirect effect was found where harsh parenting (a) increased bullying via increased irritable opposition, (b) increased victimization via increased irritable opposition, and (c) increased bullying–victimization via increased irritable opposition. Table 4 displays these indirect effects. For girls, no significant indirect effects were found.

We then computed the difference between the three indirect pathways for boys and girls and bootstrapped the differences 10,000 times with bias-corrected confidence intervals. The indirect pathways did not significantly differ for boys and girls. This indicates that similar indirect pathways are at work for boys and girls, although the pathways for the girls failed to reach significance.

Step 3. Moderation of Indirect Effects by Genotype Variant

We tested the degree to which the indirect effects (i.e., harsh parenting on bullying, victimization, and bullying–victimization, via irritable opposition) are stronger for carriers of the *MAOA* low-activity allele compared to those without.

The results for boys showed that (a) the indirect effect on bullying was not significantly moderated by *MAOA* (B = −.02; 95% bootstrapped bias-corrected CI [−.22 to .18]), (b) victimization was not significantly moderated by *MAOA* (B = −.01; 95% bootstrapped bias-corrected CI [−.14 to .20]) (see Figure 3), and (c) bullying–victimization was not significantly moderated by *MAOA* (B = −.01; 95% bootstrapped bias-corrected CI [−.21 to .18]).

Table 3. Regression model predicting peer bullying, victimization, and bullying–victimization by sex of the child at age 8

	Boys						Girls					
	Bul (8 years)		Vic (8 years)		Bul-vic (8 years)		Bul (8 years)		Vic (8 years)		Bul-vic (8 years)	
	B	*p*	B	*p*	B	*p*	B	*p*	B	*p*	B	*p*
Block 1												
MAOA (*low-activity*)	.00	.96	.00	.94	−.02	.43	−.02	.42	.01	.78	−.01	.72
Harsh (2–4 years)	.11	<.01	.12	<.01	.11	<.01	.03	.02	.08	<.01	.12	<.01
Block 2												
MAOA (*low-activity*) × Harsh	−.06	.24	−.08	.06±	−.08	.08	−.02	.43	−.02	0.59	−.03	.40

Note. Coefficients are presented in unstandardized form. MAOA = monoamine oxidase A; harsh = harsh parenting; bul = bullying; vic = victimization; bul-vic = bullying–victimization. MAOA is coded as 0 = *high-activity* hemizygotes (boys) and homozygotes (girls); 1 = *low-activity* allele carriers (boys and girls). ±Trend, falling close to significance and later tested using simple slopes analysis.

Table 4. Pathway estimates of harsh parenting and irritable opposition on bullying, victimization, and bullying–victimization by sex of the child

	Boys 95% CI					Girls 95% CI			
	Pathway estimates (B)	Low	High	*p*		Pathway estimates (B)	Low	High	*p*
Bullying									
Harsh to irrit	.39	.29	.49	<.01	Harsh to irrit	.36	.26	.46	<.01
Irrit to bully	.06	.04	.09	<.01	Irrit to bully	.02	.00	.05	>.05
Harsh to bully	.08	.03	.13	<.01	Harsh to bully	.02	.00	.05	>.05
Indirect	.03	.01	.04	—	Indirect	.01	.00	.01	—
Victimization									
Harsh to irrit	.39	.30	.49	<.01	Harsh to irrit	.35	.26	.45	<.05
Irrit to victim	.05	.03	.07	<.01	Irrit to victim	.03	.00	.05	<.01
Harsh to victim	.13	.06	.15	<.01	Harsh to victim	.07	.03	.11	<.01
Indirect	.02	.01	.03	—	Indirect	.01	.00	.02	—
Bullying–victimization									
Harsh to irrit	.39	.29	.49	<.01	Harsh to irrit	.35	.26	.45	<.01
Irrit to bully-vic	.07	.04	.09	<.01	Irrit to bully-vic	.03	.00	.05	<.05
Harsh to bully-vic	.10	.06	.15	<.01	Harsh to bully-vic	.05	.02	.08	<.01
Indirect	.02	.02	.04	—	Indirect	.01	.00	.02	—

Note. Coefficients are presented in unstandardized form. Harsh = harsh parenting; irrit = irritable opposition; bully = bullying; victim = victimization; bully-vic = bullying–victimization; indirect = indirect effect.

The results for girls showed that (a) the indirect effect on bullying was not significantly moderated by *MAOA* (B = .04; 95% bootstrapped bias-corrected CI [−.16 to .13]), (b) victimization was not significantly moderated by *MAOA* (B = .04; 95% bootstrapped bias-corrected CI [−.15 to .24]), and (c) bullying–victimization was not significantly moderated by *MAOA* (B = 0.04; 95% bootstrapped bias-corrected CI [−.16 to .24]).

That is, for boys and girls all of these indirect effects were not significantly increased or decreased when the children belonged to either genotype-variant group.

Discussion

The current study tested the extent to which *MAOA* might confer vulnerability to the relationship of harsh parenting to subsequent bullying, victimization, and bullying–victimization via increased irritable opposition. Against expectation, the results showed that there was no significant G×E between harsh parenting and outcomes of bullying and of bullying–victimization. For victimization—although we had no specific hypothesis—the G×E for boys was found to be close to significance ($p = .06$). Simple slopes indicated that *MAOA* low-activity carriers were at

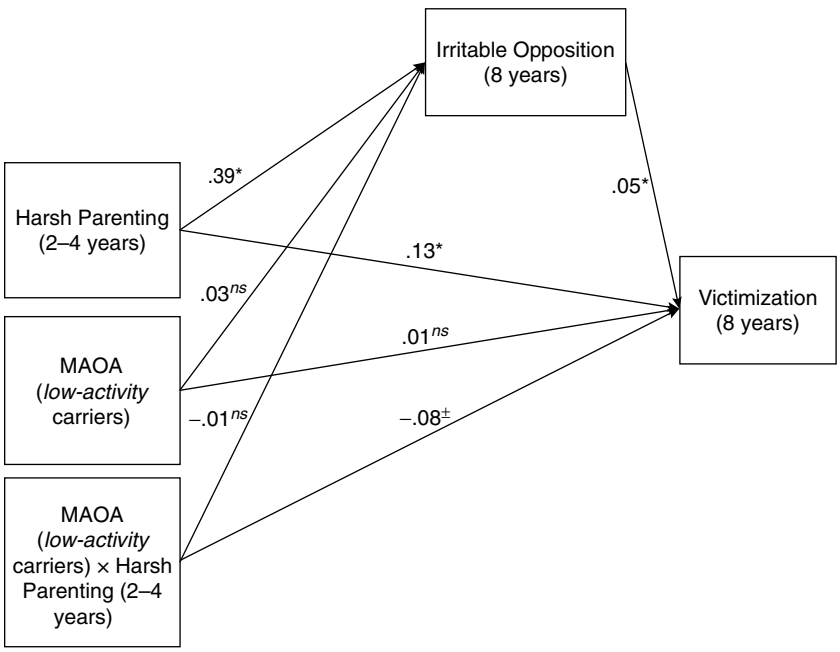

Figure 3. Boys' MAOA is coded as 0 = *high-activity* hemizygotes and 1 = *low-activity* hemizygotes. Coefficients are presented in unstandardized form. *$p < 0.01$; ± = trend; *ns* = nonsignificant. The path predicting victimization from monoamine oxidase A (*MAOA*) × Harsh Parenting was close to significance ($p = .06$). Harsh parenting and irritable opposition are parent-rated and bullying is self-rated.

higher risk of victimization than were high-activity-only carriers, although victimization levels were increased for all *MAOA* alleles across higher levels of harsh parenting. In addition, although we did not find genetic moderation of the indirect effects, for boys, but not girls, harsh parenting increased bullying, victimization, and bullying–victimization via increased irritable opposition.

First, although the low activity MAOA allele was found to increase the relationship between harsh parenting and peer victimization in boys, the result was only close to significance. What could account for this borderline significant result? The outcome of prior *MAOA* G×E studies has focused on the perpetration of aggressive behaviors but not specifically on being the victim of aggressive behaviors. Therefore, further studies examining the outcome of victimization are required in order to determine whether this result will be replicated. For victimization, a more promising endophenotype (i.e., a measure that is closer to the gene functioning than is victimization) might be one that reduces the likelihood of being victimized by others. For example, a popular, confident, self-assured, narcissistic type of temperament—which can also associate with the instrumental use of physical force (e.g., Fanti & Kimonis, 2012)—might be more appropriate. For children who are more instrumental in their delivery of aggression, the use of aggression in the home by parents could influence this type of problem-solving behavior with peers (e.g., Barker, Oliver, Viding, Salekin, & Maughan, 2011), and this would relate less to peer victimization (Fanti, Frick, & Georgiou, 2009; Salmivalli & Nieminen, 2002). In addition, our findings counter previous research that has shown greater aggression toward others by carriers of the low-activity allele who have experienced harsh parenting (e.g., Caspi et al., 2002; Kim-Cohen et al., 2006; Widom & Brzustowicz, 2006)—we did not find such an effect for bullying. We note that our bullying measure is not composed purely of physical aggression but also contains elements of relational forms of aggression such as stealing the belongings of others and calling others names, and detection of G×E has been shown to vary by how violence (including harsh parenting) in childhood is measured (Uher & McGuffin, 2010). Hence, *MAOA* may be more specific to the perpetration of physically aggressive acts than other forms of aggressive behavior.

Second, although *MAOA* did not moderate the indirect pathway, we found that, for boys, the link of early-life harsh parenting to bullying, bullying–victimization, and victimization was indirectly related to harsh parenting via an increase in childhood irritable oppositional behaviors. These findings are developmentally meaningful in that they demonstrate that, for boys, social adversity in the family relates to bullying, victimization,

and bullying–victimization through its association with child characteristics. This finding supports research on child characteristics (e.g., irritable opposition) that appear to elicit more punitive and coercive discipline practices (Bates, Pettit, & Dodge, 1995, Bates, Pettit, Dodge, & Ridge, 1998; Patterson, Reid, & Dishion, 1992). Having experienced social adversity in the family, boys may go on to model familial patterns of interaction (Patterson, DeBaryshe, & Ramsey, 1989). Hence, the present results support existing ideas with regard to the early developmental circumstances of a child who is characterized by an overly reactive hot-tempered and emotionally dysregulated behavioral pattern (Olweus, 1978, Schwartz, Dodge, Petit, & Bates, 1997) and is at high risk for both bullying behaviors toward, as well as victimization by, peers (i.e., the bully, the victim, and the bully–victim).

Remember that, for girls, although the indirect pathways failed to reach significance, the pathways still did not differ from the boys'. This may suggest that similar developmental processes are at work. It also may suggest that alternative mediating variables between harsh parenting and the study outcomes exist for girls, such as a more confident, self-assured, narcissistic type of temperament (e.g., Fanti & Kimonis, 2012). As females have been found to engage in relational (i.e., nonaggressive, socially influential) bullying (Crick & Grotpeter, 1995), it may also be worth examining this form of bullying in more depth and testing whether girls who are parented harshly develop a hostile attribution bias while becoming more adept at perspective taking and displaying social influence over time in an effort to avoid being punished.

Third, as stated, this indirect pathway was not moderated (i.e., it did not vary) by *MAOA*. That is, in none of the study outcomes did the the effects of harsh parenting on children who were carriers of low-activity *MAOA* differ via increased irritable opposition. As already mentioned when the result of *MAOA* increasing the relationship between harsh parenting and peer victimization was discussed, a more promising endophenotype might need to be examined. For example a popular, confident, self-assured, narcissistic type of temperament—that can also associate with the instrumental use of physical force (e.g., Fanti & Kimonis, 2012)—might be a worthy candidate.

Strengths and Limitations of This Study

Although this study has a number of strengths—such as its large sample size, broad scope, and longitudinal focus—a number of limitations warrant mentioning. First, the measures were brief and could have benefitted

from more detail. For example, more extensive and reliable forms of harsh parenting that include measures of psychological abuse were unavailable and would be worth examining in future studies. Second, although multiple-rater reports were used, harsh parenting was based on maternal reports, and irritable opposition was based on on maternal reports (alongside teacher reports), which raises the possibility of shared method variance. Third, as this study is correlational in nature, it does not allow for tests of causal inference, and, in addition, significant effect sizes found are not large. Fourth, although ALSPAC children and mothers represent a broad spectrum of socioeconomic status, the sample was stratified to exclude those from non-White ethnic backgrounds. Thus, the present results will benefit from replication with stratification across more ethnically diverse samples. Fifth, as with most longitudinal cohorts, attrition has occurred in the ALSPAC over time. For example, as expected, younger and more socially disadvantaged mothers were more likely to be lost to follow-up. As these predictors of attrition also predict childhood psychopathology, our sample is likely to underrepresent the most severely affected children. Of note, a ALSPAC cohort study showed that, although attrition affected prevalence, rates of antisocial behavior, and related disorders, associations between risks and outcomes remained, though conservative estimates of the likely true effects (Wolke et al., 2009).

In sum, the results of this study suggest that being a low-activity carrier of *MAOA* can increase the risk of victimization somewhat following harsh parenting more greatly in boys than can being a high-activity-only carrier. The exact mechanisms (or endophenotype) underlying this decrease in risk are unknown, but we hypothesize that they may relate to the more instrumental use of aggression (rather than reactive use of aggression). Research will need to both replicate the present results and test for endophenotypes of this kind.

References

Barker, E. D., Arseneault, L., Brendgen, M., Fontaine, N., & Maughan, B. (2008). Joint development of bullying and victimization in adolescence: Relations to delinquency and self-harm. *Journal of the American Academy of Child & Adolescent Psychiatry, 47*(9), 1030–1038. doi:10.1097/CHI.ObO13e31817eec98

Barker, E. D., Boivin, M., Brendgen, M., Fontaine, N., Arseneault, L., Vitaro, F., . . . Tremblay, R. E. (2008). Predictive validity and early predictors of

peer-victimization trajectories in preschool. *Archives of General Psychiatry, 65*(10), 1185–1192. doi:10.1001/archpsyc.65.10.1185

Barker, E. D., Oliver, B. R., Viding, E., Salekin, R. T., & Maughan, B. (2011). The impact of prenatal maternal risk, fearless temperament and early parenting on adolescent callous-unemotional traits: A 14-year longitudinal investigation. *Journal of Child Psychology and Psychiatry, 52*(8), 878–888. doi:10.1111/j.1469-7610.2011.02397.x

Barker, E. D., and Salekin, R. T. (2012). Irritable oppositional defiance and callous unemotional traits: Is the association partially explained by peer victimization? *Journal of Child Psychology and Psychiatry, 53*(11), 1167–1175. doi:10.1111/j.1469-7610.2012.02579.x

Barnett, J. H., Xu, K., Heron, J., Goldman, D., & Jones, P. B. (2011). Cognitive effects of genetic variation in monoamine neurotransmitter systems: A population-based study of *COMT, MAOA,* and *5HTTLPR*. *American Journal of Medical Genetics Part B: Neuropsychiatric Genetics, 156*(2), 158–166. doi:10.1002/ajmg.b.31150

Baron, R. M., & Kenny, D. A. (1986). The moderator–mediator variable distinction in social psychological research: Conceptual, strategic, and statistical considerations. *Journal of Personality and Social Psychology, 51*(6), 1173–1182. doi:10.1037/0022-3514.51.6.1173

Bates, J. E., Pettit, G. S., & Dodge, K. A. (1995). Family and child factors in stability and change in children's aggressiveness in elementary school. In J. McCord (Ed.), *Coercion and punishment in long-term perspectives* (pp. 124–138). New York: Cambridge University Press.

Bates, J. E., Pettit, G. S., Dodge, K. A., & Ridge, B. (1998). Interaction of temperamental resistance to control and restrictive parenting in the development of externalizing behavior. *Developmental Psychology, 34*(5), 982–995. doi:10.1037/0012-1649.34.5.982

Bentler, P. M., & Bonett, D. G. (1980). Significance tests and goodness of fit in the analysis of covariance structures. *Psychological Bulletin, 88*(3), 588–606. doi:10.1037/0033-2909.88.3.588

Boulton, M. J., & Smith, P. K. (1994). Bully/victim problems in middle-school children: Stability, self-perceived competence, peer perceptions and peer acceptance. *British Journal of Developmental Psychology, 12*(3), 315–329. doi:10.1111/j.2044-835X.1994.tb00637.x

Boyd, A., Golding, J., MacLeod, J. A., Lawlor, D., Fraser, A., Henderson, J., . . . Davey Smith, G. (2012). Cohort profile: The 'Children of the 90s'—The index offspring of the Avon Longitudinal Study of Parents and Children (ALSPAC). *International Journal of Epidemiology, 42*(1)111–127. doi:10.1093/ije/dy0s064

Browne, M. W., & Cudeck, R. (1993). Alternative ways of assessing model fit. In K. A. Bollen & J. S. Long (Eds.), *Testing structural equation models* (pp. 136–162). Newbury Park, CA: Sage.

Camodeca, M., Goossens, Frits, A., Meerum Terwogt, M., & Schuengel, C. (2002). Bullying and victimization among school-age children: Stability and links to proactive and reactive aggression. *Social Development, 11*(3), 332–345. doi:10.1111/1467-9507.00203

Caspi, A., McClay, J., Moffitt, T. E., Mill, J., Martin, J., Craig, I. W., . . . Poulton, R. (2002). Role of genotype in the cycle of violence in maltreated children. *Science, 297*(5582), 851–854. doi:10.1126/science.1072290

Collins, L. M., Graham J. W., & Flaherty B. P. (1998). An alternative framework for defining mediation. *Multivariate Behavioral Research, 33*(2), 295–312. doi:10.1207/s15327906mbr3302_5

Crick, N. R., & Grotpeter, J. K. (1995). Relational aggression, gender, and social-psychological adjustment. *Child Development, 66*(3), 710–722.

Dodge, K. A. (2006). Translational science in action: Hostile attributional style and the development of aggressive behavior problems. *Development and Psychopathology, 18*(3), 791–814. doi:10.1017/S0954579406060391

Dodge, K. A., Greenberg, M. T., Malone, P. S., & the Conduct Problems Prevention Research Group. (2008). Testing an idealized dynamic cascade model of the development of serious violence in adolescence. *Child Development, 79*(6), 1907–1927. doi:10.1111/j.1467-8624.2008.01233.x

Dodge, K. A., Lansford, J. E., Burks, V. S., Bates, J. E., Pettit, G. S., Fontaine, R., & Price, J. M. (2003). Peer rejection and social information-processing factors in the development of aggressive behavior problems in children. *Child Development, 74*(2), 374–393.

Dodge, K. A., & Pettit, G. S. (2003). A biopsychosocial model of the development of chronic conduct problems in adolescence. *Developmental Psychology, 39*(2), 349–371. doi:10.1037/0012-1649.39.2.349

Enoch, M. A., Steer, C. D., Newman, T. K., Gibson, N., & Goldman, D. (2010). The impact of early life stress *MAOA,* and gene-environment interaction on behavioral disinhibition in children. *Genes, Brain and Behavior, 9*(1), 65–74. doi:10.1111/j.1601-183X.2009.00535.x

Fanti, K. A., Frick, P. J., & Georgiou, S. (2009). Linking callous-unemotional traits to instrumental and non-instrumental forms of aggression. *Journal of Psychopathology and Behavioral Assessment, 31*(4), 285–298. doi:10.1007/s10862-008-9111-3

Fanti, K. A., & Kimonis, E. R. (2012). Bullying and victimization: The role of conduct problems and psychopathic traits. *Journal of Research on Adolescence, 22*(4), 617–631. doi:10.1111/j.1532-7795.2012.00809.x

Ford, J. D., Racusin, R., Ellis, C. G., Daviss, W. B., Reiser, J., Fleischer, A., . . . Thomas, J. (2000). Child maltreatment, other trauma exposure, and post-traumatic symptomatology among children with oppositional defiant and attention deficit hyperactivity disorders. *Child Maltreatment, 5*(3), 205–217. doi:10.1177/1077559500005003001

Fraser, A., Macdonald-Wallis, C., Tilling, K., Boyd, A., Golding, J., Davey Smith, G., . . . Lawlor, D. A. (2012). Cohort profile: The Avon Longitudinal Study of Parents and Children—ALSPAC mothers cohort. *International Journal of Epidemiology, 42*(1), 1–14. doi:10.1093/ije/dys066

Goodman, A., Heiervang, E., Collishaw, S., & Goodman, R. (2011). The 'DAWBA bands' as an ordered-categorical measure of child mental health: Description and validation in British and Norwegian samples. *Social Psychiatry and Psychiatric Epidemiology, 46*(6), 521–532. doi:10.1007/s00127-010-0219-x

Hosser, D., Raddatz, S., & Windzio, M. (2007). Child maltreatment, revictimization, and violent behavior. *Violence and Victims, 22*(3), 318–333. doi:10.1891/088667007780842829

Huang, Y. Y., Cate, S. P., Battistuzzi, C., Oquendo, M. A., Brent, D., & Mann, J. J. (2004). An association between a functional polymorphism in the monoamine oxidase A gene promoter, impulsive traits and early abuse experiences. *Neuropsychopharmacology, 29*(8),1498–1505. doi:10.1038/sj.npp.1300455

Jaffee, S. R., Caspi, A., Moffitt, T. E., Dodge, K. A., Rutter, M., Taylor, A., . . . Tully, L. A. (2005). Nature × nurture: Genetic vulnerabilities interact with physical maltreatment to promote conduct problems. *Development and Psychopathology, 17*(1), 67–84. doi:10.1017/S0954579405050042

Kim-Cohen, J., Caspi, A., Taylor, A., Williams, B., Newcombe, R., Craig, I. W., . . . Moffitt T. E. (2006). MAOA, maltreatment, and gene-environment interaction predicting children's mental health: New evidence and a meta-analysis. *Molecular Psychiatry, 11*(10), 903–913. doi:10.1038/sj.mp.4001851

MacKinnon, D. P. (2000). Contrasts in multiple mediator models. In J. Rose, L. Chassin, C. C. Presson, & S. J. Sherman (Eds.), *Multivariate applications in substance use research: New methods for new questions* (pp. 141–160). Mahwah, NJ: Erlbaum.

Manuck, S. B., Flory, J. D., Ferrell, R. E., Mann, J. J., & Muldoon, M. F. (2000). A regulatory polymorphism of the monoamine oxidase—A gene may be associated with variability in aggression, impulsivity, and central nervous system serotonergic responsivity. *Psychiatry Research, 95*(1), 9–23. doi:10.1016/S0165-1781(00)00162-1

Meltzer, H., Gatward, R., Goodman, R., & Ford, F. (2000). *Mental health of children and adolescents in Great Britain*. London: Stationery Office.

Meyer-Lindenberg, A., Buckholtz, J. W., Kolachana, B., Hariri, A. R., Pezawas, L., Blasi, G., . . . Weinberger, D. R. (2006). Neural mechanisms of genetic risk for impulsivity and violence in humans. *Proceedings of the National Academy of Sciences, USA, 103*(16), 6269–6274. doi:10.1073/pnas.0511311103

Muthén, L. K., & Muthén, B. O. (1998–2010). *M*plus: *Statistical analyses with latent variables—User's guide* (6th ed.). Los Angeles: Muthén & Muthén.

Olweus, D. (1978). *Aggression in the schools: Bullies and whipping boys*. Washington, DC, Hemisphere Press.

Patterson, G. R., DeBaryshe, B. D., & Ramsey, E. (1989). A developmental perspective on antisocial behavior. *American Psychologist, 44*(2), 329–335. doi:10.1037//0003-066X.44.2.329

Patterson, G. R., Reid, J. B., & Dishion, T. J. (1992). *Antisocial boys*. Eugene, OR: Castalia.

Pellegrini, A. D., Bartini, M., & Brooks, F. (1999). School bullies, victims, and aggressive victims. Factors relating to group affiliation and victimization in early adolescence. *Journal of Educational Psychology, 91*(2), 216–224. doi:10.1037/0022-0663.91.2.216

Preacher, I. J., & Hayes, A. F. (2008). Asymptotic and resampling strategies for assessing and comparing indirect effects in multiple mediator models. *Behavior Research Methods, 40*(3), 879–891. doi:10.3758/BRM.40.3.879

Preacher, K. J., Rucker, D. D., & Hayes, A. F. (2007). Addressing moderated mediation hypotheses: Theory, methods, and prescriptions. *Multivariate Behavioral Research, 42*(1), 185–227. doi:10.1080/00273170701341316

Sabol, S. Z, Hu, S., & Hamer, D. (1998). A functional polymorphism in the monoamine oxidase A gene promoter. *Human Genetics, 103*(3), 273–279. doi:10.1007/s004390050816

Salmivalli, C., & Nieminen, E. (2002). Proactive and reactive aggression among school bullies, victims, and bully-victims. *Aggressive Behavior, 28*(1), 30–44. doi:10.1002/ab.90004.abs

Schreier, A., Wolke, D., Thomas, K., Horwood, J., Hollis, C., Gunnell, D., . . . Harrison, G. (2009). Prospective study of peer victimization in childhood and psychotic symptoms in a nonclinical population at age 12 years. *Archives of General Psychiatry, 66*(5), 527–536. doi:10.1001/archgenpsychiatry.2009.23

Schwartz, D., Dodge, K. A., Pettit, G. S., & Bates, J. E. (1997). The early socialization of aggressive victims of bullying. *Child Development, 68*(4), 665–675. doi:10.2307/1132117

Schwartz, D., McFadyen-Ketchum, S. A., Dodge, K. A., Pettit, G. S., & Bates, J. E. (1998). Peer group victimization as a predictor of children's behavior problems at home and in school. *Development and Psychopathology, 10*(1), 87–99. doi:10.1017/S095457949800131X

Shrout, P. E., & Bolger, N. (2002). Mediation in experimental and nonexperimental studies: New procedures and recommendations. *Psychological Methods, 7*(4), 422–445. doi:10.1037//1082-989X.7.4.422

Stringaris, A., & Goodman, R. (2009). Three dimensions of oppositionality in youth. *Journal of Child Psychology and Psychiatry, 50*(3), 216–223. doi:10.1111/j.1469-7610.2008.01989.x

Toblin, R. L., Schwartz, D., Hopmeyer Gorman, A., & Abou-ezzeddine, T. (2005). Social–cognitive and behavioral attributes of aggressive victims of bullying. *Applied Developmental Psychology, 26*(3), 329–346. doi:10.1016/j.appdev.2005.02.004

Uher, R., & McGuffin, P. (2010). The moderation by the serotonin transporter gene of environmental adversity in the etiology of depression: 2009 update. *Molecular Psychiatry, 15*(1), 18–22. doi:10.1038/mp.2009.123

Widom, C. S., & Brzustowicz, L. M. (2006). MAOA and the "cycle of violence": Childhood abuse and neglect, MAOA genotype, and risk for violent and antisocial behavior. *Biological Psychiatry, 60*(7), 684–689. doi:10.1016/j.biopsych.2006.03.039

Wolke, D., Waylen, A., Samara, M., Steer, C., Goodman, R., Ford, T., . . . Lamberts, K. (2009). Selective drop-out in longitudinal studies and non-biased prediction of behaviour disorders. *British Journal of Psychiatry, 195*(3), 249–256. doi:10.1192/bjp.bp.108.053751